INTRODUCTION
At the beginning of the Christian era, population of the world was about 250 million which rose to one billion by 1930 and three billion by 1960. The present world population is about 4 billion and at the present rate of growth, may become 8 billion by the year 2000 A.D. and 16 billion by 2025 A.D. Hackett (1973) believes that the earth cannot sustain more than 10 billion people. Thus the greatest challenge faced by the modern world is the population explosion. Survival of mankind lies in retarding the growth of population.

Right from the ancient times attempts have been made to regulate the fertility of human beings. Historically the earlier methods as mentioned in ancient literature like papyrus were exotic, bizarre and unscientific (for reference see Himes, 1936). With the pioneering work carried out by Sanger, scientific attempts to regulate the human fertility were really initiated. A number of techniques were tried but a breakthrough in the control of fertility by hormones was made by Pincus (1955). He introduced the use of various synthetic analogues of progesterone and estrogens orally for the regulation of fertility of human females. These analogues included norethynodrel and norethindrone as progestagens and ethinyl estradiol and mestranol as estrogens. Rapid strides of progress were made due to the work at various drug houses and a number of formulations of steroid
contraceptives were marketed in the mid-sixties, the latest in the series being long acting contraceptives. Even though there are now numerous other methods of contraception available, the 'pill' is still the most popular method. In fact their popularity has increased with time. A report of National Institute of Health shows that the use of steroid contraceptive in U.S.A. increased by 10.3 percent within five years, 1965-1970, (Corfman, 1974).

Unluckily, use of steroid contraceptives is not free from various undesirable side effects. It has been shown that carbohydrate and lipid metabolism is deranged. It was observed that in some women using steroid contraceptives, glucose tolerance was decreased (for reference see Spellacy, 1969). But the deleterious effects on lipid metabolism were much more widespread (de Alvarez, 1973). In fact it has been put forward by Stokes and Wynn (1971) that progestin type of contraceptives effect the cholesterol metabolism and the mixed type contraceptives lead to hypertriglyceridaemia. Results show that the use of steroid contraceptives indeed increased the risk of thromboembolism and stroke (Corfman, 1974).

Hyperlipidaemia is also an important risk factor in the etiology of atherosclerosis manifesting mostly as coronary heart disease (Gordon and Kannel, 1971).

Various workers have attempted to study the mechanisms involved in the induction of hypertriglyceridaemia
by steroid contraceptives. It has been suggested that as steroid contraceptives increase the levels of blood insulin, effects on triglycerides may be secondary to 'hyperinsulinism'. However, it has been shown that in pregnancy as well as under the influence of exogenous progestins, peripheral resistance to insulin takes place (Beck and Denver, 1969; Beck and Wells, 1969; Kalkhoff et al. 1970) and 'hyperinsulinism' might be the physiological response of this increase in the resistance to the action of insulin. Further studies have shown that there is a decrease in plasma levels of PHIA in women using steroid contraceptives (Hazsard et al. 1969 a,b and Rossner et al. 1971) but at the same time utilization of fat was not decreased (Rossner et al. 1971). In fact, a recent study, surprisingly, showed an accelerated fat clearance (Kissenbah et al. 1973), and suggested that hypertriglyceridaemia may be due to an increase in the hepatic synthesis and transport of triglycerides. But such a contention has yet to be confirmed by direct experimental evidence. It has also been suggested that a decrease in the levels of PHIA may be due to some sort of 'competition' between heparin and steroid contraceptives (Hazsard et al. 1972). Even this hypothesis has yet to be supported by direct evidence. Furthermore, this hypothesis does not take into account all the aspects of the effects of steroid
contraceptives on the lipid metabolism in general and that of glycerides in particular.

Obviously mechanism of hypertriglyceridaemia cannot be studied clinically and an animal model is necessary. Earlier studies on laboratory animals treated with steroid contraceptives have been primarily designed to study the relationship of diet on the lipids (Tabacchi and Kirksey, 1973), mechanism of derangement of cholesterol metabolism (Aftergood and Alfin-Slatter, 1968, 1971) and the inter-relationship of the effects of carbohydrate and lipid metabolism (Schillinger and Gerhards, 1973). The present study was, therefore, planned to have an animal model of hypertriglyceridaemia induced by steroid contraceptives and to underline the biochemical basis of this derangement.

Findings revealed that unlike female guinea pigs, female rats when treated with mixed type of steroid contraceptives developed frank hypertriglyceridaemia. Differential studies to investigate TG synthesis, its transport from liver to peripheral blood, TG utilization and lipoprotein lipase have also been carried out.